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Sleep apnea prevalence in acute myocardial infarction — The Sleep Apnea in Post-acute Myocardial Infarction Patients (SAPAMI) Study

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ABSTRACT

Background: While sleep apnea (SA) might be a modifiable cardiovascular risk factor, recent data suggest that SA is severely underdiagnosed in patients after acute myocardial infarction (MI). There is limited evidence about day–night variation of onset of MI on dependence of having SA. We therefore investigated the prevalence of SA and examined the day–night variation of onset of MI in acute MI patients.
Methods: We prospectively studied 782 consecutive patients admitted to the hospital with the diagnosis of acute MI. All subjects underwent sleep evaluations using a portable device after at least 48 h post-admission. Using the apnea–hypopnea index (AHI), groups were defined as patients without SA (<5 events/h), mild SA (5–15 events/h), moderate SA (15–30 events/h), and severe SA (≥30 events/h).
Results: Almost all patients (98%) underwent urgent coronary angiography and 91% of patients underwent primary PCI. Using a threshold of AHI≥5 events/h, SA was present in 65.7% of patients after acute MI. Mild SA was present in 32.6%, moderate in 20.4% and severe in 12.7%. The day–night variation in the onset of MI in all groups of SA patients was similar to that observed in non-SA patients. From 6 AM to 12 PM, the frequency of MI was higher in both SA and non-SA patients, as compared to the interval from 12 AM to 6 AM (all $p < 0.05$).
Conclusion: There is a high prevalence of SA in patients presenting with acute MI. Peak time of MI onset in SA patients was between 6 AM and noon, similar to that in the general population. Whether diagnosis and treatment of SA after MI will significantly improve outcomes in these patients remains to be determined.

1. Introduction

Sleep apnea (SA) is highly prevalent in patients with cardiovascular disease and is thought to contribute to the onset and progression of cardiac and vascular damage [1–3]. Patients with SA are often obese, with an increased risk for diabetes and hypertension. These co-morbidities, in the setting of apnea, repetitive hypoxemia, reflex sympathetic activation, and blood pressure surges may predispose to acute myocardial infarction (MI) [4].

Several studies have suggested that patients with acute MI have a high likelihood of SA with estimates ranging from 50 to 66% [2,3,5–7]. Existing studies have been limited by relatively small sample sizes ranging from 12 to 105 subjects [2,3,5–7]. Given the heterogeneity of the acute MI population, particularly with regard to variables such as drug therapy, body habitus, gender and age, it is important that large sample sizes be studied in order to obtain a more accurate assessment of the prevalence of SA in this patient population.

In the general population, the onset of MI exhibits a diurnal periodicity that peaks between 6 AM and 12 PM [8]. Beta blockade has been shown to blunt the early morning peak in MI [9]. Furthermore, we have shown that in patients with obstructive sleep apnea (OSA) the peak occurrence of MI

Abbreviations: ACE, angiotensin converting enzyme; ADP, adenosine diphosphate; AHI, apnea–hypopnea index; BMI, body mass index; CI, confidence interval; CPAP, continuous positive airway pressure; CSA, central sleep apnea; DBP, diastolic blood pressure; eGFR MDRD, estimated glomerular filtration rate using modification of diet in renal disease formula; LAD, left anterior descending; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; OSA, obstructive sleep apnea; PCI, percutaneous coronary interventions; RCA, right coronary artery; LCX, circumflex branch of left coronary artery; RIM, ramus intermedius; SA, sleep apnea; SBP, systolic blood pressure; STEMI, ST elevation myocardial infarction.

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is higher between 12 AM and 6 AM [10]. However, in a study that included 40 middle-aged men, the peak of MI onset was reported to be between 6 AM and 12 PM in those with SA [11].

Therefore, we conducted a prospective study of all patients presenting to our hospitals with acute MI to further investigate the prevalence of sleep apnea in patients with MI, and the potential effects of sleep apnea on the day–night variation of acute MI.

2. Methods

This study was conducted in accordance with the amended Declaration of Helsinki and was approved by the Ethics Committees of the University Hospital Brno, and St. Anne's University Hospital, Brno, and all patients provided informed consent. The study was conducted between January 2010 and June 2012 at the Department of Internal Medicine and Cardiology, University Hospital Brno, Czech Republic and between January 2011 and June 2012 at the Department of Internal Medicine and Cardioangiology, St. Anne's University Hospital, Brno, Czech Republic.

2.1. Study population

We prospectively studied 782 patients admitted to both hospitals with the diagnosis of acute MI. The diagnostic criteria for acute MI were based on those established by the Czech Society of Cardiology [12,13]. These criteria are in compliance with similar recommendations of the European Society of Cardiology (ESC), the American College of Cardiology (ACC) and American Heart Association (AHA). Although consecutive patients were eligible, recruitment was based on exclusion criteria listed below and on patients consenting to participate. The exclusion criteria were: age below 18, uncertain time of onset of MI (onset of symptoms [ischemic chest discomfort or equivalent such as shortness of breath] reported by patients), and previous continuous positive airway pressure (CPAP) therapy. Both centers are tertiary institutions and have catheterization laboratories available 24 h a day throughout the year.

All patients underwent a complete clinical (history and physical examination) and biochemical evaluation including assessment of high-sensitivity cardiac troponin T (Roche Diagnostic) and electrocardiogram at the time of admission. Two additional measurements of high-sensitivity cardiac troponin T were obtained at intervals of 12 h. Patients were monitored and treated according to common standards of acute myocardial infarction management [12,13]. Therapy at admission, at the time of the sleep study, and at discharge, was recorded.

Transthoracic echocardiography was performed (GE Vivid7 [General Electric Company] with an S4 probe) using standard projections. Echocardiographic measures were based on the average value of three measurements. The disk summation method (modified Simpson's rule) was used for the assessment of left ventricular ejection fraction (LVEF).

All coronary angiography and percutaneous coronary interventions (PCI) were digitally acquired and analyzed by an investigator blinded to other characteristics of the patient. A stenosis greater than 50% was considered significant.

2.2. Sleep study

All subjects underwent sleep evaluations using a portable diagnostic device [Apnealink™ (ResMed)] after at least 48 h post-admission, provided that they were in stable condition (without the need for oxygen therapy, invasive or non-invasive artificial pulmonary ventilation, intra-aortic balloon counterpulsation, mechanical heart support, hemodialysis, intravenous vasodilators, inotropes, or intravenous diuretics, and without any symptoms of airway inflammation or symptoms of acute exacerbation of chronic obstructive pulmonary disease or asthma). Drugs such as hypnotics, narcotics or other medications which may affect breathing were prohibited during sleep study. The ApneaLink™ records a patient's nasal respiratory pressure, and pulse oximetry during sleep. Outputs from the ApneaLink™ were analyzed manually by three investigators blinded to the patient's clinical characteristics. Respiratory events were classified according to the standard criteria of the American Academy of Sleep Medicine [14]. The apnea–hypopnea index (AHI) was calculated by dividing the number of events (apneas and hypopneas) by the number of hours of sleep. Onset of sleep was recorded at the time at which respiration settled to a rhythmic stable pattern. The end of the recording time was the waking time reported by subjects. Using the apnea–hypopnea index (AHI), groups were defined as patients without SA (AHI < 5 events/h), mild SA (5 to 15 events/h), moderate SA (15 to 30 events/h), and severe SA (>30 events/h). We did not differentiate between OSA and central sleep apnea (CSA).

The device registered and derived the number of apneas and hypopneas, apnea–hypopnea index, respiratory index, apnea index, hypopnea index, oxygen desaturation index, baseline, minimal and average oxygen saturation, durations when oxygen saturation was lower than 90, 85 and 80%, minimal, maximal and average heart rate, average respiratory rate, and number of snoring episodes.

ApneaLink™ has been validated against conventional polysomnography and found to be an accurate instrument for the detection of apneas, hypopneas, snoring, and oxygen desaturations. A number of studies report high sensitivity and specificity at a variety of thresholds for defining sleep disordered breathing [15–19].

2.3. Statistical analysis

Personal data security was guaranteed by assigning each patient in the study a trial number. All data were recorded in electronic form, and processed in the Biostatistics Department of the International Clinical Research Center, Brno, using standard descriptive statistics (mean, standard deviation, median and its 95% confidence interval, minimum and maximum for continuous variables, frequency tables for categorical variables). For the assessment of differences between groups of patients for continuous variables, the Kruskal–Wallis and Mann–Whitney tests were used for non-normal distributions. Categorical variable interrelationships were determined by Chi-square test. Two-tailed p values<0.05 were considered as significant, and were corrected for multiple testing. Statistical analyses were performed using software package SAS 9.2.

3. Results

We studied 782 patients. Of these, 175 (22.4%) patients had technically inadequate limited sleep studies (less than 4 h recording time or inability to score study due to excessive artifact). We therefore analyzed the data from 607 patients who had good quality sleep study records. The majority of patients (98%) underwent urgent coronary angiography and 91% of patients underwent primary PCI. The remaining patients were treated conservatively or underwent elective coronary artery by-pass grafting. None of the patients had received fibrinolytic therapy. The median duration from hospital admission to overnight sleep study was 4 days (range 2 to 14 days). Using a threshold of AHI≥5 events/h, SA was present in 65.7% of patients after acute MI. Mild SA was present in 32.6%, moderate in 20.4% and severe in 12.7% of patients (Fig. 1).

Patients' baseline characteristics are shown in Tables 1–5. Data are shown for all subjects, as well as those without SA, and with mild, moderate or severe SA. There were statistically significant differences between the groups in age, body mass index (BMI), proportion of

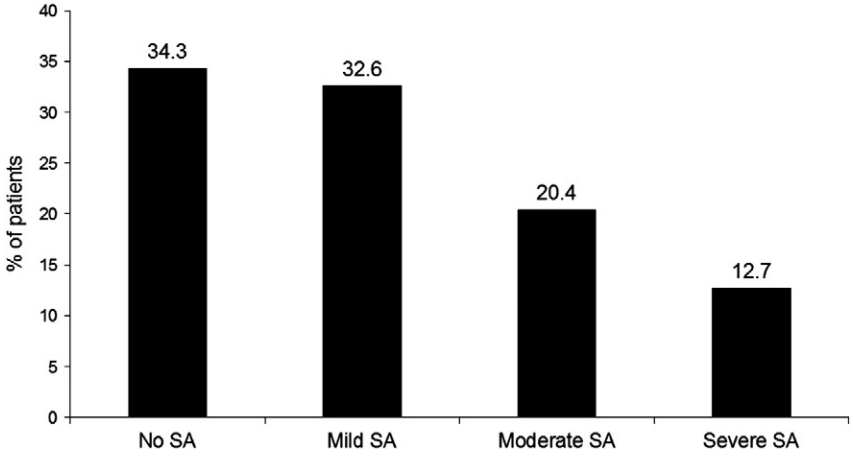

Fig. 1. Prevalence of sleep apnea in patients after acute myocardial infarction.

Table 1
Patient characteristics at the time of myocardial infarction according to sleep apnea categories (continuous parameters).

Parameter	Overall (n = 607)	No SA (n = 208)	Mild SA (n = 198)	Moderate SA (n = 124)	Severe SA (n = 77)	p-Value[a]
Age [years]	62.0 (61.00–63.00)	59.0 (57.00–61.00)	63.0 (61.00–65.00)	64.5 (61.00–67.00)	67.0 (62.00–70.00)	<0.001
BMI [kg/m^2]	27.9 (27.68–28.40)	27.1 (26.10–27.70)	28.4 (27.70–29.10)	29.0 (27.80–29.60)	28.7 (27.00–30.10)	0.007
SBP [mm Hg]	140.0 (136.00–140.00)	140.0 (130.00–140.00)	140.0 (135.00–145.00)	140.0 (135.00–150.00)	140.0 (130.00–150.00)	0.609
DBP [mm Hg]	80.0 (80.00–80.00)	80.0 (80.00–80.00)	80.0 (80.00–80.00)	80.0 (80.00–85.00)	80.0 (80.00–80.00)	0.562
LV EF [%]	50.0 (50.00–52.50)	50.5 (49.00–55.00)	54.5 (50.00–55.00)	50.0 (45.00–53.00)	45.0 (42.00–49.00)	<0.001

Values are presented as median (95% CI).
[a] p-Value of Kruskal–Wallis test.

Table 2
Patient characteristics at the time of myocardial infarction according to sleep apnea categories (categorical parameters).

Parameter	Category	Overall (n = 607)	No SA (n = 208)	Mild SA (n = 198)	Moderate SA (n = 124)	Severe SA (n = 77)	p-Value[a]
Gender	Male	73.5	68.8	69.7	81.5	83.1	0.009
	Female	26.5	31.3	30.3	18.5	16.9	
Type of myocardial infarction	STEMI	70.0	81.7	64.6	66.1	58.4	<0.001
	NSTEMI	30.0	18.3	35.4	33.9	41.6	
Culprit lesion	LAD	43.0	52.9	37.4	36.3	41.6	0.051
	RCX	21.1	18.8	25.8	20.2	16.9	
	RCA	32.9	26.0	34.8	38.7	37.7	
	RIM	0.3	0.5	0.5	0	0	
	Unknown	2.6	1.9	1.5	4.8	3.9	
Single vessel disease	Yes	37.2	41.8	36.9	34.7	29.9	0.259
Two vessel disease	Yes	31.6	29.8	34.3	33.1	27.3	0.618
Multi vessel disease	Yes	27.8	24.5	25.8	29.8	39.0	0.087
PCI	Yes	90.8	93.8	92.4	87.9	83.1	0.024
Killip class	1	85.8	86.1	90.9	82.3	77.9	0.222
	2	10.2	9.6	6.6	12.9	16.9	
	3	3.1	3.4	2.0	3.2	5.2	
	4	0.8	1.0	0.5	1.6	0	
NYHA class	1	79.3	79.3	82.8	77.3	75.0	0.278
	1.5	2.8	2.3		7.6	2.3	
	2	12.8	14.9	10.8	10.6	15.9	
	2.5	3.1	1.1	4.3	1.5	6.8	
	3	1.7	1.1	2.2	3.0		
	3.5	0.3	1.1				

Values are presented as percentage.
[a] p-Value of Chi-square test.

men, proportion of non-ST elevation MI, history of hypertension, dyslipidemia, diabetes mellitus type 2, coronary artery disease, prior MI, stroke/transient ischemic attack, peripheral artery disease, and also medications being taken prior to admission for myocardial infarction (ACE inhibitors, angiotensin receptor blockers, aspirin, statins). All of these were increased with increasing severity of SA. There were also statistically significant differences in LVEF, and cholesterol levels, which decreased with increasing severity of SA. Patient with moderate and severe SA were less likely to undergo PCI. However, in further gender analysis, all of these were found to be significant only in men. Only age was significantly different with increasing SA severity in women. The vast majority of participants were receiving beta blockers and ACE inhibitors or angiotensin receptor blockers at the time of sleep study (Table 6).

The day–night variation in the onset of MI in all groups of SA patients was similar to that observed in non-SA patients (Fig. 2). From 6 AM to

Table 3
Patient comorbidities.

Parameter	Category	Overall (n = 607)	No SA (n = 208)	Mild SA (n = 198)	Moderate SA (n = 124)	Severe SA (n = 77)	p-Value[a]
Hypertension	Yes	58.5	49.0	56.1	64.5	80.5	<0.001
Dyslipidemia	Yes	23.7	19.2	20.2	28.2	37.7	0.004
Diabetes mellitus type 2	Yes	25.5	20.7	20.7	33.9	37.7	0.001
Known coronary artery disease	Yes	22.4	17.8	20.2	25.8	35.1	0.012
Prior myocardial infarction	Yes	15.8	10.6	13.6	20.2	28.6	0.001
Congestive heart failure	Yes	2.3	1.4	2.5	3.2	2.6	0.747
Chronic obstructive pulmonary disease	Yes	5.4	5.8	5.6	3.2	7.8	0.560
Chronic kidney disease	Yes	3.3	3.8	3.0	2.4	3.9	0.890
Stroke/transient ischemic attack	Yes	8.9	5.8	9.1	6.5	20.8	<0.001
Peripheral artery disease — lower limb	Yes	5.1	2.4	4.0	5.6	14.3	<0.001
Atrial fibrillation	Yes	5.6	4.3	5.1	5.6	10.4	0.252
Smoking status	Current	40.5	45.8	40.2	35.5	35.5	0.312
	Former	19.8	17.4	19.6	25.6	17.1	
	Never	39.7	36.8	40.2	38.8	47.4	

Values are presented as percentage.
[a] p-Value of Chi-square test.

Table 4
Laboratory characteristics.

Parameter	Overall (n = 607)	No SA (n = 208)	Mild SA (n = 198)	Moderate SA (n = 124)	Severe SA (n = 77)	p-Value[a]
Cholesterol [mmol/l]	5.0 (4.90–5.11)	5.1 (4.90–5.30)	5.1 (4.90–5.40)	4.9 (4.70–5.30)	4.4 (4.10–4.90)	0.005
HDL cholesterol [mmol/l]	1.1 (1.06–1.10)	1.1 (1.02–1.10)	1.1 (1.01–1.12)	1.1 (1.10–1.20)	1.0 (0.92–1.10)	0.116
LDL cholesterol [mmol/l]	3.0 (3.00–3.11)	3.0 (2.90–3.20)	3.1 (3.00–3.30)	3.0 (2.70–3.30)	2.7 (2.30–3.00)	0.057
Triglycerides [mmol/l]	1.5 (1.46–1.63)	1.5 (1.42–1.70)	1.6 (1.49–1.77)	1.4 (1.22–1.63)	1.4 (1.15–1.69)	0.086
Fasting glycemia [umol/l]	6.2 (6.10–6.40)	6.3 (6.10–6.40)	6.1 (5.90–6.40)	6.1 (5.80–6.40)	6.7 (6.10–7.60)	0.117
eGFR MDRD [ml/min]	82.4 (79.85–85.01)	85.4 (81.13–91.21)	82.3 (78.09–86.35)	81.6 (78.07–87.60)	76.9 (66.84–86.13)	0.08
Peak troponin T [ug/l]	1.3 (0.97–1.58)	1.4 (0.93–1.88)	1.1 (0.80–1.68)	1.4 (0.66–1.85)	1.2 (0.55–2.88)	0.855
Hemoglobin [g/l]	142.0 (141.00–143.00)	142.0 (139.00–143.00)	144.0 (140.00–146.00)	142.5 (140.00–146.00)	140.5 (137.00–145.00)	0.566

Values are presented as median (95% CI).
[a] p-Value of Kruskal–Wallis test.

Table 5
Medications at the time of myocardial infarction.

Parameter	Overall (n = 607)	No SA (n = 208)	Mild SA (n = 198)	Moderate SA (n = 124)	Severe SA (n = 77)	p-Value[a]
ACE inhibitors	34.2	27.4	32.8	42.3	42.9	0.014
Angiotensin receptors blockers	12.4	7.7	14.6	11.4	20.8	0.016
Beta-blockers	30.4	25.5	31.8	33.3	35.1	0.277
Aspirin	24.3	19.2	23.7	26.8	35.1	0.042
ADP receptor blockers	4.6	4.3	2.5	8.1	5.2	0.138
Statins	22.3	16.8	20.7	22.0	41.6	<0.001
Calcium channels blockers — dihydropyridine type	21.8	19.7	20.7	24.4	26.0	0.584
Calcium channels blockers — verapamil	2.3	1.9	0.5	4.1	5.2	0.057
Diuretics	23.6	18.3	24.2	26.8	31.2	0.090

Values are presented as percentage.
[a] p-Value of Chi-square test.

Table 6
Medications at the time of sleep study.

Parameter	Overall (n = 607)	No SA (n = 208)	Mild SA (n = 198)	Moderate SA (n = 124)	Severe SA (n = 77)	p-Value[a]
ACE inhibitors	84.9	84.0	83.3	90.2	83.1	0.327
Angiotensin receptors blockers	7.1	4.9	7.6	5.7	14.3	0.046
Beta-blockers	93.5	93.2	94.9	92.7	92.2	0.785
Aspirin	94.2	97.6	97.5	90.2	83.1	<0.001
ADP receptor blockers	93.5	98.5	93.9	91.9	81.8	<0.001
Statins	97.2	98.1	98.0	95.1	96.1	0.351
Calcium channels blockers — dihydropyridine type	13.1	12.6	13.1	13.8	13.0	0.992
Calcium channels blockers — verapamil	0.5	0.5	0.5	0.8	0	0.889
Diuretics	34.5	27.2	31.8	41.9	49.4	0.001

Values are presented as percentage.
[a] p-Value of Chi-square test.

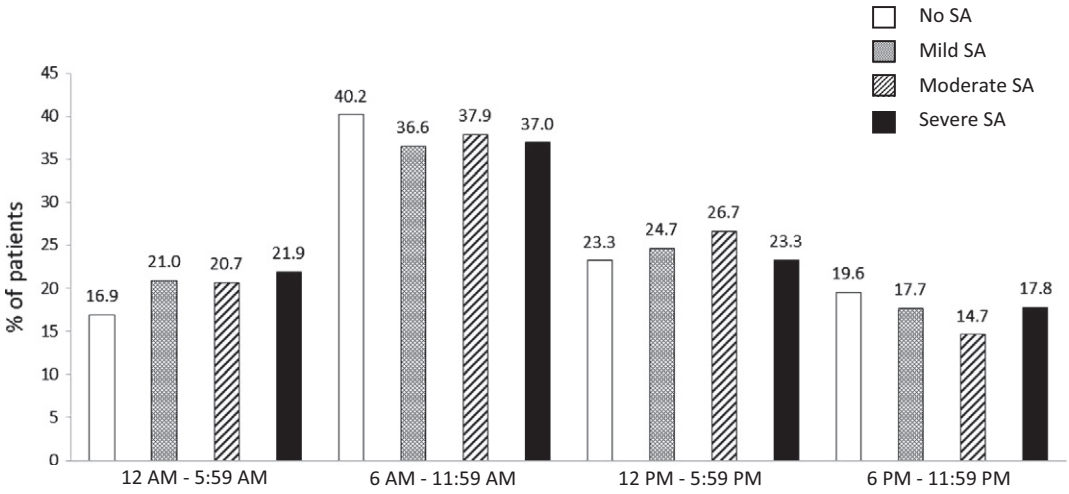

Fig. 2. 6-hour epochs of MI occurrence.

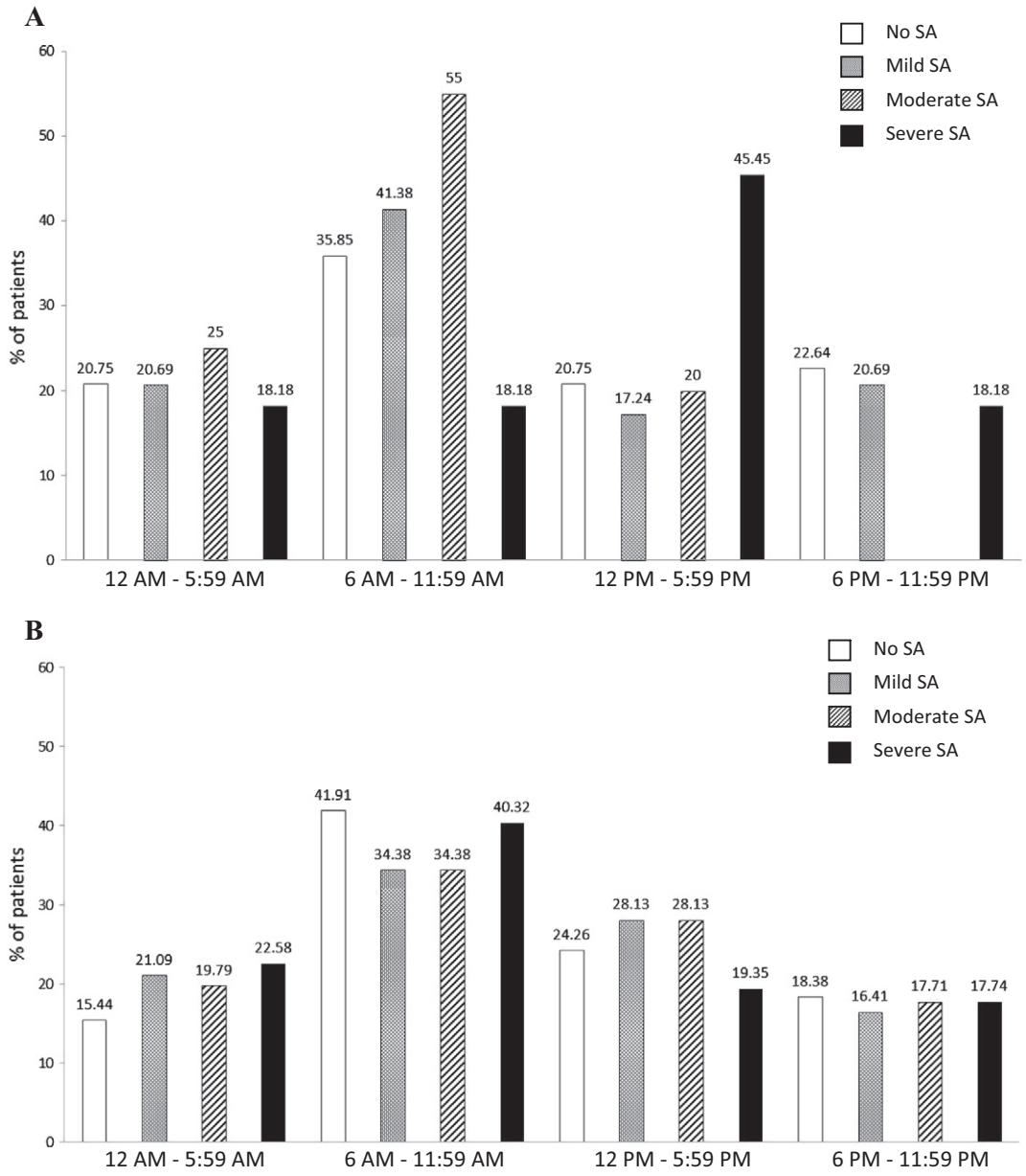

Fig. 3. 6-hour epochs of MI occurrence according to gender — females are shown in upper panel (A) and males are shown in lower panel (B).

12 PM, the frequency of MI was higher in both SA and non-SA patients, as compared to the interval from 12 AM to 6 AM (all $p < 0.05$). In men, the likelihood of nocturnal MI (between 12 AM and 6 AM) was greater as sleep apnea severity increased ($p = 0.06$) (Fig. 3). We also found no evidence of any effect of beta-blocker therapy on the timing of MI.

4. Discussion

We found a high prevalence of previously undiagnosed SA in acute MI patients, and that patients with SA have diurnal variation in MI occurrence similar to that observed in patients without SA. Several studies have suggested that patients with acute MI have a high likelihood of SA. However, with the exception of two studies [3,7], all consisted of a heterogeneous study population including unstable angina, MI, and heart failure [2,5,6]. Prior studies also used varying thresholds for the diagnosis of SA, and none of them divided patients into groups according to severity of SA, likely because of the relatively small sample sizes [2,3,5–7]. In our study, using a threshold of AHI≥5 events/h, we found a relatively high prevalence of SA (65.7%). A threshold of AHI≥10 events/h resulted in a slightly lower prevalence of SA (49.6%) than in other studies [2,6]. Using a threshold of AHI≥15 events/h, we found a relatively lower prevalence of SA (33.1%) [3,5,20]. Since patients with known SA, previously treated by CPAP, were not included in our study, it is probable that we might have slightly underestimated the actual prevalence of SA in acute myocardial infarction patients. On the other hand, the number of patients who are treated for SA in the Czech Republic is still minimal.

Differences in diagnostic criteria, types of diagnostic devices, timing of sleep studies, sample sizes, and characteristics of the study population may all affect the determination and prevalence of SA. While SA might be a modifiable cardiovascular risk factor, recent data suggest that SA is severely underdiagnosed in patients after acute MI [21]. Deficiencies in diagnosis and hence therapy may be due to logistic and economic considerations. The gold standard for diagnosing SA remains overnight polysomnography, which is expensive and not widely available [22]. Discharging acute myocardial infarction patients from the hospital without a definitive diagnosis of sleep apnea would likely

delay appropriate intervention. Therefore, sleep evaluation using portable diagnostic devices may provide a useful and economical strategy for identifying patients at risk for SA and maybe improving prognosis [23–25].

To our knowledge, this is the first study to show that screening for SA is relatively simple and effective, and can be easily implemented into the diagnostic evaluation of all acute myocardial infarction patients. However, the optimal timing of a sleep study in patients after acute myocardial infarction is still unresolved. Furthermore, whether treatment of SA in patients after MI will actually improve outcomes remains unknown.

Our findings regarding the timing of MI onset in SA versus non-SA patients, differ from our previous report which suggested that OSA may reverse the usual day–night variation of myocardial infarction and shift the timing of MI from the morning hours to the night [9]. However, in the present study, we observed that men with severe SA may be more likely to have nocturnal MI. The higher likelihood of nocturnal MI onset in men with the most severe SA is consistent with the construct of OSA-induced acute sympathetic, pressor [26], hypoxemic and prothrombotic stress [27] resulting in the initiation of cardiac ischemia [28–30], acute coronary syndrome, fatal arrhythmia [31] or sudden death [32].

The difference in our results regarding the overall peak occurrence of MI in SA patients may be explained, at least in part, by the following factors. Our current study included a substantially greater number of subjects, but from a different geographical region (Czech Republic). The patients included in the current study had lower BMI, higher blood pressures, and were more likely to be smokers. In addition, this was a relatively selected group with a high (>90%) likelihood of undergoing primary PCI. The time of occurrence of MI was extracted from the patient records, and we cannot rule out the development of intermittent chest discomfort or anginal equivalent earlier during the night. The use of portable monitoring of oxygen saturation and nasal airflow in our current study, versus complete overnight polysomnography, may affect the characterization and quantification of sleep apnea, and does not enable differentiation between obstructive and central apnea. However, based on other studies in a similar population, we can reasonably assume that the vast majority of these patients had OSA [33]. In addition, the timing of sleep evaluation (4 days after MI versus 17 days after MI in the prior study) and the use of analgesics, sedatives and hypnotics, may have influenced the sleep evaluation. Finally, the 22% of sleep evaluations that were technically inadequate could have accounted in part for the differences between this and our prior study.

The timing of the sleep diagnostic test could be a concern since previous studies indicated that SDB is temporarily worsened in the acute phase after an MI and may be transient [5,7]. However another study suggested that the prevalence of SA shortly after an MI is the same as that recorded six months later [6]. In our study every subject underwent sleep study after at least 48 h post-admission, and all were in stable condition.

Important strengths of this study include, first, the sample size of 607 patients, approximately six-fold greater than any of the previous studies. Second, all consecutive patients with acute MI in the city of Brno (approximately 400,000 inhabitants), who consented to participate, were prospectively studied, thus providing a representative prevalence estimate in the general population.

5. Conclusion

In summary, we found a high prevalence of previously undiagnosed SA in acute myocardial infarction patients. In this population, peak occurrence of MI onset was between 6 AM and noon in SA patients, similar to what is observed in the general population. Considering that this is a group at high risk for heart failure, fatal arrhythmia, recurrent MI and sudden death, treatment of SA may provide an important strategy for secondary prevention. Whether treating SA reduces post-MI morbidity and mortality awaits the findings of randomized controlled trials.

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Disclosures

Dr. Somers serves as a consultant for Respicardia, ResMed, Neu Pro, Sorin Inc, and Price Waterhouse Coopers, and works with Mayo Health Solutions and their industry partners on intellectual property related to sleep and cardiovascular disease (CZ.1.07/2.3.00/20.0022). Dr. Kuniyoshi is a full time employee of Philips Respironics.

Dr. Ludka had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. All authors have contributed significantly to the work and have reviewed and approved the final version of the manuscript.

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